Risks and Consequences of Nuclear Weapons Accidents in South Asia

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Abstract

We analyze the dispersal of plutonium into the atmosphere and consequent biological hazards from nuclear weapon accidents. Such accidents involving nuclear weapons could be caused, for example, by missile and jet fuel fires and explosions — and have occurred on a number of occasions to US nuclear weapons. We use the Wedge Model of aerosol dispersal to estimate the amount of plutonium that would be inhaled by a surrounding population and the resulting radiological damage in the form of increased cancer fatalities in the event of such an accident. Our results suggest that such an accident in or near a large South Asian city could lead to about 5000 cancer deaths, and perhaps four times as many. In the event of an accident at a military base at some distance from a medium-sized city, there could be 200 - 800 cancer deaths. Therefore, prudence would dictate that India and Pakistan not deploy nuclear weapons or store them close to aircraft or ballistic missiles. Keeping the weapons disassembled would further reduce the risk of accidental detonation.

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1. Introduction

Despite having carried out a number of nuclear weapons tests in May 1998, neither India nor Pakistan are believed to have yet deployed their nuclear weapons. Apart from command and control issues, deployment carries a number of dangers, including those associated with accidents involving assembled nuclear weapons, with the risks increasing when the weapons are deployed with their delivery systems, and further increased where the weapons systems are kept on a high state of alert. These potential dangers include in order of increasing seriousness:

- Possible burning of the high explosive in the weapons leading to melting of the fissile material core;
- Detonation of the explosive leading to aerosolisation of part of the fissile material and its airborne dispersal;
- An explosion that produces a nuclear yield.

This paper analyses these risks of such accidents, focusing especially on the intermediate case involving the dispersal of plutonium in the event of a non-nuclear explosion, and the health and environmental consequences of such an accident. Briefly, section 2 describes the experience of nuclear weapons accidents in the nuclear weapon states, especially the United States, and assesses similar risks in South Asia. Section 3 models airborne dispersal of plutonium, and this is followed in Section 4 by a survey of health impacts of plutonium inhalation. These results are applied to hypothetical accident scenarios in South Asia in Section 5. A brief discussion of the results and their implications is presented in Section 6.

2. Past Accidents

Ever since the beginning of the nuclear age, there have been accidents involving nuclear weapons and their delivery vehicles. Information on such accidents is scarce and it is not possible to list all of them. It is claimed that there have been at least 230 nuclear

weapons accidents involving the U.S., U.S.S.R., and the U.K. between 1950 and 1980; in addition, there were many more near misses.[1]

The United States has the longest experience of deploying nuclear weapons with its armed forces, and is the only country to have used them. It is also the country which has released the most information about its nuclear weapons. Between 1945-1951 the US nuclear arsenal consisted of aircraft delivered fission bombs, which were designed so that the nuclear capsule had to be manually inserted. This was done while the aircraft was in flight, and removed before landing. Starting in 1952, the warheads were designed to permit the capsules to be inserted mechanically, allowing aircraft to carry bombs externally and the development of ballistic missile warheads. It was only in the mid-1950s that the US developed "sealed pits" which allowed the weapons to remain assembled at all times, with the fissile material pit enclosed inside the high explosive lenses. This allowed for reductions in weapon size and weight and increased operational readiness. These designs however increased the risk of accidental detonation of the high explosive in the weapons leading to either the dispersal of the fissile material or a nuclear explosion.[2]

These risks were recognised and attempts made over the years to reduce the likelihood of such accidents. Despite these attempts, accidents continued to occur. An official summary released by the U.S. Department of Defense in 1981 lists 32 accidents involving U.S. nuclear weapons between 1950 and 1980. This includes a number of instances where the high explosive in nuclear weapons has burned or detonated and led to contamination.[3] Table 1 (overleaf) lists those U.S. accidents in the summary reported as leading to contamination.

[1] See for example Shaun Gregory and Alistair Edwards, The Hidden Cost of Deterrence: Nuclear Weapons Accidents 1950-1988, *Bulletin of Peace Proposals*, vol. 20, no. 1, 1989, pp. 3-26

[2] Sidney Drell and Bob Peurifoy, "Technical Issues of a Nuclear Test Ban," *Annual Reviews of Nuclear and Particle Science*, vol. 44, 1994, pp.285-327.

[3] U.S. Department of Defense in coordination with Department of Energy, *Narrative Summaries of Accidents Involving U.S. Nuclear Weapons, 1950-1980 (Interim)*, 1981.

Table 1: List of U.S. Nuclear Weapon Accidents Involving Plutonium Dispersal

Date	Location	Type of accident	HE Burn	HE Detonation
05/22/57	Kirtland Air Force Base (AFB)	inadvertent jettison		X
01/31/58	SAC base overseas	taxi exercise, fire	X	
11/06/58	Dyess AFB	crash on takeoff		X
07/06/59	Barkesdale AFB	crash on takeoff, fire	X	
06/07/60	McGuire AFB	missile fire	X	
11/13/63	Medina Base[4]	in storage	X	X
12/08/64	Bunker Hill AFB	taxi crash, fire	X	
10/11/65	Wright-Patterson AFB[5]	transport aircraft fire on ground		
01/17/66	Palomares, Spain	mid-air collision, crash		X
01/21/68	Thule Greenland	crash after abandonment		X

Source: Sidney Drell and Bob Peurifoy, Technical Issues of a Nuclear Test Ban, *Annual Reviews of Nuclear and particle Science, vol. 44, 1994, pp. 285-327* (based on U.S. DOD Narrative History)

These accidents typically involve delivery vehicles, either aircraft or missiles. Most notable among missile accidents is the 1960 accident involving a U.S. BOMARC missile at the McGuire Air Force base in New Jersey which suffered an explosion and a fire involving the missile's fuel tanks.[6] The significance of this accident is that it happened when the missile was in a "ready storage" condition (permitting launch in two minutes).[7] A related example is the September 1980 Titan II ICBM fuel explosion at Damascus, Arkansas, which did not, however, result in plutonium dispersal.[8]

[4] This accident occurred when components from obsolete weapons were being dismantled.
[5] This accident only involved nuclear weapons components and a dummy training unit. There was no HE detonation or burn; nevertheless, it resulted in some plutonium contamination with "minimal radiation hazard."
[6] Jaya Tiwari and Cleve J. Gray, "U.S. Nuclear Weapons Accidents," available on the internet at: http://www.cdi.org/Issues/NukeAccidents/accidents.htm
[7] U.S. Department of Defense in coordination with Department of Energy, *Narrative Summaries of Accidents Involving U.S. Nuclear Weapons, 1950-1980 (Interim).*
[8] "U.S. Nuclear Weapons Accidents: Danger In Our Midst," *The Defence Monitor*, Vol. X, No. 5, 1981, Center for Defence Information, Washington D.C.

There have been many other accidents involving missiles and missile silos, fortunately less severe. The U.S. Air Force has revealed that in a period of four years, between 1975 and 1979, there were 125 accidents at its missiles sites, and a further ten from March 1979 to September 1980.[9]

There have also been accidents involving aircraft, the most famous being over Palomares, Spain, and near Thule, Greenland. In both cases, aircraft carrying nuclear weapons crashed and the high explosive surrounding the nuclear core detonated. This led to dispersal of plutonium over a large region.[10]

There have been accidents involving U.S. naval nuclear weapons as well. One assessment lists 383 accidents between 1945 and 1988.[11] These have included a number of instances in which nuclear weapons were lost at sea as a result of the sinking of submarines and ships.

Information about accidents in the erstwhile Soviet Union is harder to obtain, but one source lists over 25 serious nuclear weapon accidents there.[12] These included a 1977 accident in which, reportedly, fuel leaked from a nuclear missile in its silo and subsequently exploded.[13] Such accidents continue to happen. A recent example is the ballistic missile explosion at Vladivostok that occurred as the missile was being unloaded from a transport ship on June 16, 2000.[14] According to preliminary information the missile caught on the pier railing, which led to a leak of approximately 3 tons of the oxidizing agent. A number of people were injured and villages had to be evacuated.

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[9] "U.S. Nuclear Weapons Accidents: Danger In Our Midst," *The Defence Monitor*.

[10] In the case of Palomares, 2.26 km^2 were contaminated with high plutonium ground concentrations, in excess of 11.8 kBq/m^2. E. Iranzo, S. Salvador and C. E. Iranzo, "Air Concentrations of ^{239}Pu and ^{240}Pu and Potential Radiation Doses to Persons Living near Pu-contaminated Areas in Palomares, Spain," *Health Physics* Vol. 52, No. 4, April 1987, pp. 453-461.

[11] William Arkin and Joshua Handler, "Naval Accidents 1945-1988," Neptune Paper No. 3, Greenpeace and The Institute for Policy Studies, Washington, D.C., 1989, p. 79.

[12] Shaun Gregory, *The Hidden Cost of Deterrence: Nuclear Weapons Accidents* (London: Brassey's, 1990) pp. 184-190.

[13] Gregory, *The Hidden Cost of Deterrence: Nuclear Weapons Accidents*, p. 188.

[14] British Broadcasting Corporation, "Toxic cloud moves along Russian Far Eastern coast after missile fuel leak," June 16, 2000

South Asia

While neither India nor Pakistan are believed to have deployed nuclear weapons as yet, it is worth noting that India's Draft Nuclear Doctrine calls for a "triad of aircraft, mobile land-based missiles and sea-based assets," and for them to be configured "for rapid punitive response."[15] Pakistan has issued no comparable doctrine but it is likely that it too will deploy its weapons.

If India and Pakistan deploy their nuclear weapons, they too shall face the risk of accidents involving nuclear weapons and their delivery systems. Experience suggests that there may be significant risks of aircraft and missile accidents. India's Comptroller and Auditor General reported in 1997 that there had been 187 accidents and 2729 "incidents" involving Indian Air Force (IAF) aircraft between April 1991 and March 1997, in which the IAF lost 147 aircraft and 63 pilots.[16] Data on Pakistan Air Force (PAF) accidents are less easily available. According to the Pakistan Institute for Air Defence Studies, there were 11 major PAF accidents between January 1997 and August 1998.[17]

Both India and Pakistan have been developing and testing a number of ballistic missiles. There have been no reports of accidents involving ballistic missiles in South Asia. But experience elsewhere suggests accidents are certainly possible. Of particular concern are the liquid fuelled missiles, India's Prithvi and Pakistan's Ghauri, which may have significant risks.

India's Prithvi missile is fuelled by a liquid propellant. According to most reports the oxidizer is inhibited red fuming nitric acid (IRFNA)[18] and the fuel is a 50:50

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[15] National Security Advisory Board, "Draft Nuclear Doctrine," http://www.indiagov.org/govt/indnucld.htm.

[16] Report of the Comptroller and Auditor General of India on the Ministry of Defence for the year ended March 1997, http://www.cagindia.org/reports/defence/1998_book1/index.htm.

[17] Ayaz Ahmad Khan, "Air Accidents In Spite of High Efficiency," *Defence Journal* (August 1998) http://www.defencejournal.com/aug98/airaccidents.htm.

[18] The most common type of nitric acid used as an oxidizer is RFNA (*red fuming nitric acid*) which consists of concentrated nitric acid that contains 5 to 20% dissolved nitrogen dioxide. Since nitric acid has a high specific gravity (1.5 to 1.6), it allows for a smaller missile. Compared to concentrated nitric acid (also called *white fuming nitric acid*) RFNA is more energetic, stable in storage and less corrosive to many tank materials. In order to make this even less corrosive, a little Hydrogen Flouride or some other flouride ion is added, which causes a flouride layer to form on the wall of the container, thus inhibiting the action of the nitric acid. This combination is called IRFNA. IRFNA gives off red-brown fumes that are poisonous and droplets on the skin cause burns and sores that do not heal readily. George P. Sutton, *Rocket*

combination of xylidine and triethylamine.[19] This combination is hypergolic, i.e., self igniting when mixed, and highly volatile and has to be loaded just prior to launch.

Pakistan's Ghauri is reportedly based on the North Korean No-Dong missile.[20] This is claimed to have a cluster of four North Korean Scud Mod B engines. The fuel is given as unsymmetrical dimethylhydrazine with inhibited red fuming nitric acid as an oxidiser.[21] There have been only a few tests of this missile.

As with missile systems elsewhere, there seem to have been problems. For example, it has been reported that during the fourth test of Prithvi, the missile engine did not fire because of faulty wiring.[22] Similarly, it is claimed that the first of three user trials by the Indian Army was postponed because of a faulty nozzle in the fuel feed mechanism, allowing fuel to leak into the engine casing.[23]

Propulsion Elements: An Introduction to the Engineering of Rockets (New York: John Wiley and Sons, 1992) p. 251.

[19] See for example Hormuz Mama, "Improved Prithvi Missile Launched," *International Defense Review*, August 1, 1992, p. 784.

[20] David Wright, "An Analysis of the Pakistani Ghauri Missile Test of April 6, 1998," *Science and Global Security*, Vol. 7, No. 2, 1998; Joseph S. Bermudez, "A History of Ballistic Missile Development in the DPRK," Monterey Institute of International Studies, Center for Non Proliferation Studies, Occasional Paper No. 2, 1999. However, it has also been suggested that Ghauri may use RP1 (Kerosene) as fuel; S. Chandrashekar, "The Origins and Antecedents of the Ghauri Missile – An Assessment," *Current Science*, Vol. 76, No. 3, February 10, 1999, pp. 280-285.

[21] David Wright and Timur Kadyshev, "An Analysis of the North Korean Nodong Missile," *Science and Global Security*, Vol. 4, (2), 1994, pp. 129-160.

[22] Raj Chengappa, *Weapons of Peace: The Secret Story of India's Quest to be a Nuclear Power*, (New Delhi: Harper Collins, 2000), p. 362 .

[23] Greg Gerardi, "India's 333rd Prithvi Missile Group," *Jane's Intelligence Review*, Vol. 7, No. 8, 1995, pp. 361-364.

3. Plutonium Dispersal Analysis

Categories of Accidents

A fire or fuel explosion near a nuclear weapon could lead to a range of consequences depending on the source and intensity of the fire, as well as the design of the weapon and its high explosive. It may be useful to classify such incidents into four categories of increasing health risk to members of the general public:

1. The high explosive (HE) in the nuclear weapon does not burn or detonate; the fissile material is unaffected.
2. The HE in the weapon catches fire and burns but does not detonate. This could lead to melting and fragmentation of the fissile material core.
3. The HE detonates and converts the fissile material into aerosol. However, there is no nuclear yield.
4. The HE detonates and causes a nuclear explosion.

An example of the first category of accidents is the 1980 Titan II Intercontinental Ballistic Missile (ICBM) explosion where the fuel tank of the missile exploded, catapulting its 9 megaton warhead about 200 meters. Fortunately, it was recovered intact.[24]

More serious because of the distinct possibility of plutonium contamination are accidents of the second category, where the fire or explosion leads to the burning of the high explosive (HE) inside the weapon. Of the 32 cases of US nuclear weapon accidents for the period 1950-1980 in the official list released by the US Department of Defence, there were 8 incidents of HE burn, half of which led to plutonium contamination.[25] An example of this was the accident at the McGuire Air Force Base, where, on June 7, 1960, a BOMARC air defense missile was destroyed when a high-pressure helium tank exploded and ruptured the missile's fuel tank. The fire burned for over 30 minutes;

[24] "U.S. Nuclear Weapons Accidents: Danger in Our Midst," *The Defence Monitor*.
[25] Drell and Peurifoy, "Technical Issues of a Nuclear Test Ban."

airmen continued to pour water on the smouldering rocket for 15 hours.[26] It is significant that the missile was reportedly in a "ready storage" condition, permitting launch in two minutes when the accident occurred.

Although safety devices prevented the HE from detonating it did burn and along with it the entire warhead containing plutonium, enriched uranium and bottled tritium. The use of water in fire-fighting led to further spread of this radioactive material, leading to contamination of the ground beyond the area beneath the molten debris. Nevertheless, the plutonium contamination was by and large limited to within about 30 meters from the explosion.

The release of plutonium into the environment carries a range of public health risks.[27] The two primary routes of exposure are ingestion and inhalation. Ingestion of plutonium is a less significant risk since almost all of the plutonium is excreted within a few days.[28] The more serious risk comes from inhalation of very small plutonium particles (a few microns in diameter), which can stay imbedded deep in the lungs typically for periods of the order of a year. In accidents like the one at the McGuire Base, where the HE did *not* detonate, the estimated amount of plutonium converted into respirable aerosol is only about 0.05 – 0.07 %.[29] Essentially all of the plutonium will remain within a hundred meters or so. Due to this small amount of aerosol and limited dispersal, accidents of the second category have limited public health impact. There is, however, a hazard to those in the immediate vicinity and those involved in decontaminating the affected area.

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[26] Joseph Gambardello, "Plutonium spill neither gone nor forgotten, 40 years later," *Philadelphia Inquirer* June 1, 2000.

[27] In case of accidents that disperse fissile material, plutonium based weapons have significantly more severe health effects when compared to those using highly enriched uranium, and we concentrate on these.

[28] The International Commission on Radiological Protection estimates that only 0.05% of ingested plutonium is absorbed by the gastrointestinal system. ICRP, *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 2 Ingestion Dose Coefficients*, ICRP Publication 67, (New York: Pergamon, 1994), p. 127.

[29] D.R.Stephens, "Source Terms for Plutonium Aerosolization from Nuclear Weapons Accidents," Lawrence Livermore National Laboratory Report UCRL-ID-119303; John M. Haschke, "Evaluation of Source-Term for Plutonium Aerosolization," Los Alamos National Laboratory Report LA-1231, 1992.

Category 3 Accidents: HE Detonation and Pu Dispersal

The third category of accidents consists of those where the HE inside the weapon actually detonates. In this case effectively all of the plutonium will be oxidised into plutonium oxide (PuO_2) and aerosolized.[30] Of this aerosol, about 20% will be particles of respirable size.[31] This aerosol will rise with the hot gases created by the explosion, mix with the air and spread. Any prevailing wind would transport it to considerable distances. The detailed distribution of the aerosol as a function of time will be complicated and depend sensitively on the specific parameters associated with the accident event. These factors include the location and power of the explosion, the winds, the terrain, rainfall, particulate matter already in the atmosphere to which the aerosol may become attached and so on.

To get an order of magnitude estimate of the dispersal of plutonium, we use a simple analytical model that has been used previously for estimating the effects of a nuclear weapons accident.[32] This is the 'Wedge Model', developed originally as part of the American Physical Society's study on light-water reactor safety.[33]

The wedge model makes some simplifying assumptions about the aerosol concentration profile. Upon explosion, the PuO_2 aerosol will rapidly mix upward within the mixing layer of the atmosphere, which typically has a height H between 300 and 2500 meters. The model assumes that this height remains constant as the plume moves downwind in a fixed direction and at a constant speed. Its crosswind spread (i.e.

[30] Stephens, "Source Terms for Plutonium Aerosolization from Nuclear Weapons Accidents".
[31] W.G. Sutcliffe, R.H. Condit, W.G. Mansfield, D.S.Meyers, D.W. Layton and P.W.Murphy, "A Perspective on the Dangers of Plutonium," Lawrence Livermore National Laboratory Report UCRL-JC-118825 (1995); Stephens, "Source Terms for Plutonium Aerosolization from Nuclear Weapons Accidents." However, in one experiment at the Nevada Test Site, it was found that 99% of aerosol plutonium particles produced by the HE-detonation of a nuclear explosive test device, found within a 16 km (10 miles) radius of ground zero, had physical diameters of less than 2.5 microns, indicating that essentially all of the fallout particles were in the respirable size range. J. L. Dick and T. P. Baker, Jr. (1967), "Operation Plumbbob—Test Group 57, Program 73: Monitoring and Decontamination Techniques for Plutonium Fallout on Large-Area Surfaces," WT- 1512, Air Force Special Weapons Command, Kirtland AFB, NM, cited in David Chanin and Walter Murfin, "Site Restoration: Estimation of Attributable Costs From Plutonium-Dispersal Accidents," Sandia National Laboratory Report, SAND96-0957, May 1996, http://plutonium-erl.actx.edu/restoration.html
[32] Steve Fetter and Frank von Hippel, "The Hazard from Plutonium Dispersal by Nuclear-warhead Accidents," *Science and Global Security*, Vol. 2, pp. 21-42, (1990).

perpendicular to the wind direction) is taken to be an arc subtending a wedge angle θ at the explosion point. This angle generally varies from 0.05 to 0.3 radians.

The entire plume will move downwind at the wind velocity *u*, and will be centered after time *t* at a distance $r = ut$ with some radial (downwind) spread *d(r)*. This geometry is illustrated in figure 1 (overleaf) where the annular segment gives the horizontal section of the plume at some instant of time *t*. Within this plume-volume of height H, angular width θ, and thickness *d(r)* the aerosol concentration ρ(*r*) is assumed to be uniform at any given time, although it will decrease with *r* because of spreading in width and deposition of the aerosol on the ground.

[33] "Report to the American Physical Society by the Study Group of Light-water Reactor Safety," *Reviews of Modern Physics* ,Vol. 47, Supplement no 1, (1975).

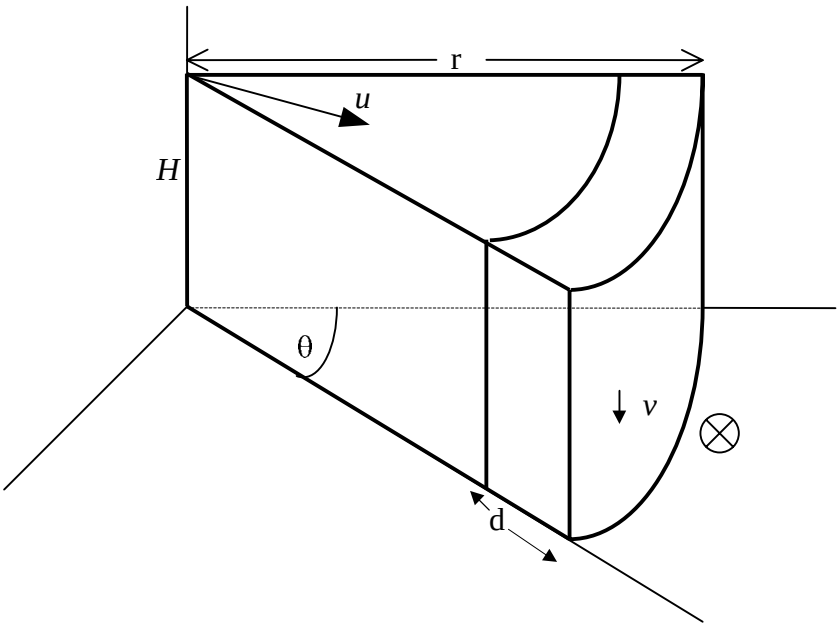

Fig .1: Schematic picture of the plume configuration at some instant of time in the wedge model. The aerosol-contaminated air is in the annular segment.

Let Q(r) be the amount of plutonium present in the cloud at distance r (i.e. after time t = r/u). Assuming that the amount deposited on the ground is proportional to the amount present in the atmosphere, we have

$$Q(r) = Q_0 e^{-r/L} \qquad (1)$$

where Q_0 is the total mass of plutonium that became aerosolised into a respirable form and the length L represents the average distance travelled by an aerosol particle before it gets deposited. Clearly

$$L = \frac{Hu}{v} \qquad (2)$$

where v is, by definition, the deposition velocity. The amount deposited per unit area is:

$$\sigma(r) = \frac{(-1)}{r\theta}\frac{dQ}{dr} = \frac{1}{rL\theta} Q_0 e^{-r/L} \qquad (3)$$

The plume has a volume $Hrd(r)\theta$, and hence the concentration is:

$$\rho(r,\theta,z) = \frac{1}{rH\theta\, d(r)} Q_0 e^{-r/L} \qquad (4)$$

Now, consider a person (shown by the cross in the figure) who is about to be immersed in the cloud. Since the plume has thickness $d(r)$ it will pass her in time $\tau = d(r)/u$. If her breathing rate is b m^3/sec, she will breathe in a volume $b\tau$ containing an amount of plutonium given (in mg) by:

$$m(r) = \frac{Q(r)}{Hrd(r)\theta}\frac{b\,d(r)}{u} = \frac{Q_0 b}{Hru\theta} e^{-r/L} \qquad (5)$$

Notice that this result is independent of the thickness $d(r)$.

There is now a considerable amount of theoretical and empirical support for the assumption that the biological risk from radiation exposure is a linear function of

radiation dose at low doses.[34] Then, if a given dose is shared among N people, the risk of cancer death per person is reduced to 1/N, but since each of N people now suffers this risk, the total probable number of cancer deaths remains the same. Thus, in the linear approximation, the probable total number of cancer deaths depends only on the total amount of plutonium inhaled by the exposed populace.

If the population density per unit area within the wedge is P(r, θ) then the total amount of plutonium inhaled by the population will be:

$$M = \int_0^{\infty} dr \int_0^{\theta} d\theta' r\, m(r)\, P(r,\theta') = \frac{Q_0 b}{Hu\theta} \int_0^{\infty} dr \int_0^{\theta} d\theta' P(r,\theta') e^{-r/L} \quad (6)$$

For cases where the population density can be taken to be uniform with P(r, θ) = P_0, this reduces to:

$$M_u = \frac{bQ_0 P_0 L}{Hu} = \frac{bP_0 Q_0}{v} \qquad (7)$$

where *v* is the deposition velocity of the aerosol. An example of such a case would be if the explosion takes place within or upwind of a large densely populated city, large enough for most of the aerosol to be deposited. This urban accident scenario would be the worst case short of a nuclear explosion.

A more realistic example may be one where a middle sized city of width *a* km which happens to lie downwind within the wedge angle at a distance R from some cantonment or airfield where the explosion occurs (see figure 2).

[34] A linear model requires that there be no minimum threshold dosage for health risks. It is believed that for cancers and other genetic effects, there is no threshold. See *Health Risks of Radon and Other Internally Deposited Alpha-Emitters (BEIR IV)* (Washington, D.C.: National Academy Press, 1988) p. 177. The ICRP also asserts that "there are no adequate grounds for assuming a real threshold" and uses a simple proportional relationship at low doses. International Committee on Radiological Protection, *1990 Recommendations of the International Committee on Radiological Protection* ICRP Publication 60, (New York: Pergamon Press, 1991) p. 18. Public health criteria in the U.S. and many other countries use such a no-threshold, linear model. There is an ongoing controversy about the validity of the linear, no-threshold model to arbitrarily low doses of radiation (see, for example, articles and letters in *Physics Today*, September 1999, April, and May 2000). However, there is less doubt about the applicability of a linear, no-threshold model for heavily ionizing (High LET) radiation such as the alpha particles emitted by plutonium decay than for lightly ionizing (Low LET) radiation such as gamma rays and beta particles.

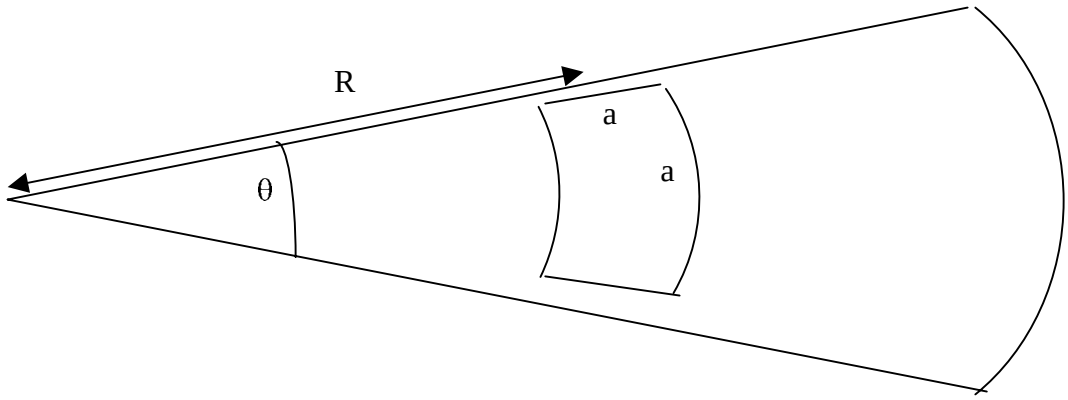

Fig 2: A schematic representation of a city of width *a* lying in the path of the plume at a distance R from an explosion.

For such a situation one goes back to general formula (6). For simplicity we will assume that the city is an annular piece at distance *R* from the explosion and subtending an angle $\theta_c = a/R$. Within the city we can approximate the population density as uniformly equal to $P_{0.}$ Then we have, for the total plutonium breathed in by that city's populace during plume passage, the result:

$$M = \frac{Q_0 b}{Hu\theta}\int_0^{\infty} dr \int_0^{\theta} d\theta' P(r,\theta') e^{-r/L}$$

$$= \frac{Q_0 b}{Hu\theta} P_0 \theta_c \int_R^{R+a} dr\, e^{-r/L}$$

$$= M_u \frac{\theta_c}{\theta}\left[e^{-R/L} - e^{-(a+R)/L}\right] \qquad (8)$$

where M_u is the urban accident case result described earlier. In addition, there would be the plutonium breathed in by the people in the surrounding countryside.

Re-suspension

The formulae derived above apply to the plutonium inhaled from the original contaminated plume from the explosion as it moves downwind. As we shall see, even if the explosion were to take place in a densely populated area, the amount inhaled during plume passage is only about 0.1 % of the total respirable plutonium aerosol content. The rest is deposited on the ground. But, as time passes, the plutonium that has been deposited on the ground could be re-suspended into the air through a variety of mechanisms like winds, clearing of debris, people walking, traffic, sweeping, eventual digging or ploughing of the land and so on. Unless the populace moves out of the entire contaminated area for a very long period of time or the area is decontaminated, they will continue to breathe in plutonium. Grazing animals, inhaling near soil level, might be particularly prone to such uptake of resuspended plutonium.[35]

This phenomenon is usually parameterised by a resuspension coefficient K, defined as the ratio of the plutonium concentration in the air to the surface density of plutonium on ground.[36] The resuspension coefficient is a function of time and the height above the ground that the concentration in air is measured. The literature lists many different functional forms to model this resuspension coefficient.[37] The leading term at some fixed height in most of these has an exponential form: $K(t) = K_0 e^{-st}$

Integrating this over time gives M_r, the amount of resuspended plutonium that is inhaled:

$$M_r = Mv\left[\left(\frac{1}{s}\right)K_0(1-e^{-st})\right]$$

where M is the plume passage inhalation amount derived earlier in equation (6), v is the deposition velocity. Long term evacuation or effective decontamination of the entire area

[35] W. J. Bair and R. C. Thompson, "Plutonium: Biomedical Research," *Science*, February 22, 1974, pp. 715-722.
[36] *Reactor Safety Study* (Washington, D.C.: U.S. Nuclear Regulatory Commission, report # NUREG-75/014, 1975), Appendix VI, pp. 8-9.
[37] L. R. Anspaugh *et al* "Resuspension and Redistribution of Plutonium in Soils," *Health Physics*, Vol. 29, October 1975, pp. 571-582; Chanin and Murfin, "Site Restoration: Estimation of Attributable Costs From Plutonium-Dispersal Accidents" (Appendix C, Tables C-1 and C-2, p. C-8) summarizes several resuspension relations.

for a large, densely populated city would be very difficult. In that case, the population would be exposed to this resuspended plutonium, for a period *t* of several decades. The parameter *s* is of the order 1 (year)$^{-1}$. Then to a very good approximation the term e^{-st} can be neglected and the above expression reduces to:

$$M_r = \frac{MvK_0}{s} \qquad (9)$$

The total amount of plutonium inhaled during plume passage and subsequent re-suspension is:

$$M_{tot} = M\left(1 + vK/s\right) \qquad (10)$$

We will later apply these formulae to estimate the amount of plutonium inhaled by the population and the consequent cancer fatality toll in hypothetical category 3 explosions in the South Asian subcontinent. The numerical values of some of the parameters that enter into the formulae are known more precisely than others. The breathing rate *b* is known fairly precisely as a function of age and of activity level. The wind velocity can vary a lot, but its value at a given place and time may be available. The total amount of plutonium will depend on the weapon, and is in principle known very precisely, but less certain is the fraction that is converted to respirable PuO_2 aerosol. A figure of 20% has been suggested as a reasonable working number – however, experiments have yielded figures ranging from 10 to nearly 100%.[38] Some of the other parameters, which can also vary considerably, fall in the following ranges:[39]

The mixing layer height H: 300 to 2500 meters

The wedge angle θ: 0.1 to 0.3 radians

The deposition velocity *v*: 0.001 to 0.1 m/sec

[38] Fetter and von Hippel, "The Hazard from Plutonium Dispersal by Nuclear-warhead Accidents"; . Sutcliffe, Condit, Mansfield, Meyers, Layton and Murphy, "A Perspective on the Dangers of Plutonium."
[39] Fetter and von Hippel, "The Hazard from Plutonium Dispersal by Nuclear-warhead Accidents" and *Reactor Safety Study*, table VI, B-1.

Following the American Physical Society study on reactor safety and Fetter and von Hippel, we choose representative values of these parameters. These will be specified when we apply these results to the South Asian example.

Notice however that the parameters that enter in equation (7), which corresponds to the uniform population density case, are relatively well defined. The main variation comes from the deposition velocity *v*, which may be as high as 0.1 m/sec if it is raining.

4. Health Impact of Exposure to Plutonium

The principal risk from exposure to plutonium is by inhalation. The typical plutonium used in nuclear weapons is mostly plutonium-239 with a half-life of 24,400 years but has small admixtures of higher isotopes, such as plutonium-240 (half-life 6,580 years) and plutonium-241 (half-life 13.2 years) coming from successive neutron captures. What is termed weapons-grade plutonium, for example, typically contains about 93.8% of Pu-239, 5.8 % of Pu-240 and 0.35% of Pu-241.[40] Each milligram of plutonium-239 corresponds to an activity in Bequerels (Bq, number of disintegrations per second) of:

$$Activity = \frac{mass}{Atomic\,weight} \times \frac{Avagadro\,number \times \ln(2)}{Halflife} = 2.3 \times 10^6 \,\frac{Bq}{mg}$$

Because of the much shorter half-lives of the higher isotopes, they would also contribute significantly to the activity. Including the contribution from the alpha decay of Pu-240 increases the specific activity to 2.65 MBq/mg.[41]

To calculate the radiation dose per Bq of inhaled plutonium, we adopt the model developed by the International Commission on Radiological Protection (ICRP), which describes the deposition of inhaled plutonium and its subsequent behaviour.[42] (See Appendix I.) Based on this model, we calculate an effective dose coefficient (EDC,

[40] J. Carson Mark, "Explosive Properties of Reactor-Grade Plutonium," *Science and Global Security*, Vol. 4, 1993, pp. 111-128.

[41] Even though Pu-241 has a much shorter half life, it is a beta emitter. As Table 2 shows, the radiation dose per unit activity of Pu-241 is much smaller and we will ignore that contribution. However, Pu-241 decays into Am-241, which *is* an alpha emitter and that would contribute about 10% of the dose after one half-life of Pu-241, namely 14.4 years, from the time of manufacture of the weapon plutonium core.

[42] We use the most recent ICRP Publication available that deals with plutonium; ICRP, *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 4 Inhalation Dose Coefficients*, ICRP Publication 71, (New York: Pergamon, 1996).

which gives the equivalent radiation dose coming from breathing in 1 Bq of the substance) for different isotopes of plutonium in different forms. The different forms of plutonium refer to their behaviour in terms of solubility. The ICRP categorizes slow absorption forms as Type S, medium absorption forms as Type M, and fast absorption forms as Type F. Oxides of plutonium formed at high temperatures, about 1000 °C, as in the case of plutonium aerosolized in explosions, are assigned to Type S. The equivalent whole-body dose coefficients to humans at different ages are given in Table 2.[43] For completeness, we also include the dose coefficient of americium-241, which results from the radioactive decay of plutonium-241. These use a quality factor or relative biological effectiveness index of 20 for alpha radiation and 1 for beta and gamma radiation.[44]

Table 2: Equivalent Whole-body Doses for Different Isotopes and Solubilities of Inhaled Plutonium and Americium

Compound	Effective Dose Coefficient at Age (Sv/Bq)		
	3 months	15 years	Adult (male)
Pu-239 (Type S)	$4.3X10^{-5}$	$1.7X10^{-5}$	$1.6X10^{-5}$
Pu-239 (Type M)	$8.0X10^{-5}$	$4.7X10^{-5}$	$5.0X10^{-5}$
Pu-239 (Type F)	$2.1X10^{-4}$	$1.1\ X10^{-4}$	$1.2\ X10^{-4}$
Pu-240 (Type S)	$4.3X10^{-5}$	$1.7X10^{-5}$	$1.6X10^{-5}$
Pu-241 (Type S)	$2.2X10^{-7}$	$1.7X10^{-7}$	$1.7X10^{-7}$
Am-241 (Type S)	$4.6X10^{-5}$	$1.7X10^{-5}$	$1.6X10^{-5}$

At the relatively low levels of plutonium inhalation that most people would be exposed to following dispersal from an accident, the primary effects of exposure to plutonium are an increased probability of cancers of the lung, liver and bone.[45] (High dose effects are described in Appendix II.) Lesser effects resulting from the inhalation of plutonium oxide include a reduction in the number of white blood cells, among the most

[43] Due to the greater solubility, plutonium of Types M and F is cleared faster from the lung and accumulates in the bone surfaces and the liver. Hence, even though the radiation dose to the lung from these types is much smaller than for Type S, the radiation dose to the whole body is much greater.
[44] International Committee on Radiological Protection, *1990 Recommendations of the International Committee on Radiological Protection* ICRP Publication 60, p. 6.
[45] *Health Risks of Radon and Other Internally Deposited Alpha-Emitters* .

radiosensitive cells in the body; chromosomal aberrations in blood lymphocytes; and in the case of rats, increased incidence of mammary tumours.[46]

Human epidemiological studies of the effects of plutonium inhalation are limited. Hence, two approaches can be used to estimate risks. The first involves the use of estimated lifetime risks obtained from laboratory animal experiments. Difficulties with this approach relate to the many differences between animals and humans, including differences in histological types of cancers, differences in confounding exposures (e.g., smoking), differences in spontaneous risks, and differences in life span. The second approach involves expressing risks obtained from humans exposed to alpha radiation from radon decay products, for which data from epidemiological studies with miners are available, or to low-LET[47] x-ray and gamma radiation in terms of dose (or dose equivalent) to the lung or other relevant tissues. These risk estimates are then applied to the doses resulting from high-LET alpha radiation from transuranic elements. One difficulty with this approach is that there may be characteristics of specific exposures that are not fully reflected in a single dose estimate but that may affect resulting health effect risks.

The International Commission on Radiological Protection has suggested using a rate of 500 fatal cancers and 100 non-fatal cancers per 10^4 person-Sv.[48] To obtain the number of cancer deaths per unit mass of inhaled plutonium (taken to be of Type S), we use the age dependence of the coefficients in Table 2 to first compute a age-weighted EDC.

The fraction of the population under the age of 15 in India is 39.3%.[49] The data for Pakistan is similar.[50] We will assume that this population is uniformly distributed over

[46] *Health Risks of Radon and Other Internally Deposited Alpha-Emitters.*

[47] The rate at which ionizing particles impart energy locally to a medium is known as the linear energy transfer, commonly abbreviated as LET. The damage to living tissue by absorption of a given amount of energy is generally greater as the distance over which this energy is imparted decreases, i.e., as the LET increases. Jacob Shapiro, *Radiation Protection: A Guide to Scientists and Physicians*, (Cambridge, U.S.A.: Harvard University Press, 1990), p.34.

[48] International Committee on Radiological Protection, *1990 Recommendations of the International Committee on Radiological Protection*, p. 22. This estimate may be fairly conservative as it ignores the large fraction of non-fatal skin and thyroid cancers.

[49] Onkar Singh and A. K. Singh, "Population Growth and Family Planning in India: An Analysis," in *Strategies in Development Planning*, edited by A.K. Singh, V. K. Rai and A. P. Mishra, (New Delhi: Deep and Deep, 1997) pp. 355-367.

the age range of 0-15 and we will linearly interpolate between the EDCs at ages 3 months and 15 years. This results in an average of 3.0 X 10^{-5} Sv/Bq for children under 15 years. Convoluting this with the population distribution of 39.3% below 15 years of age and using the adult EDC for the remaining 60.7%, we obtain an age-weighted EDC:

$$Weighted\ \ EDC = (0.393 \times 3.0 + 0.607 \times 1.6) \times 10^{-5} = 2.15 \times 10^{-5}\, Sv / Bq$$

To convert this to a dose per unit mass of inhaled plutonium, we use the figure of 2.65 MBq/mg obtained earlier to get:

$$Dose\ per\ unit\ mass = 2.15 \times 10^{-5}\, Sv / Bq \times 2.65 \times 10^{6}\, Bq / mg = 57\, Sv / mg$$

Using the ICRP risk estimate, we obtain a figure of:

$$Cancer\ deaths\ per\ unit\ \ mass = 2.85\ cancers / mg$$

There are other estimates of the number of cancer deaths per unit of radiation. The U.S. National Research Council Committee on the Biological Effects of Ionizing Radiation (BEIR) proposes that specifically for plutonium, the risk estimates are 700 lung-cancer deaths/10^4 person-Sv, 80-1100 bone-cancer deaths/10^4 person-Sv and 300 liver-cancer deaths/10^4 person-Sv on the basis of human studies.[51] This would lead to an estimate of 6-12 cancer deaths/mg of inhaled plutonium. Similarly, Fetter and von Hippel extrapolate from the risk of pulmonary neoplasia (a cancer) to beagle dogs subjected to high doses of plutonium and obtain 12 cancer deaths per milligram of weapons grade plutonium inhaled.[52] Both these estimates are significantly higher than the figure suggested by the ICRP, which we use.

5. Estimated Casualties in South Asia

We now apply the results of the wedge model and the health impact risk estimates to possible accidents involving nuclear weapons in South Asia where the HE detonates and disperses plutonium (i.e., category 3). We look at two examples.

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[50] World Resources Institute, UNEP, UNDP and World Bank, *World Resources 1998-1999* (New York: Oxford University Press, 1998), p. 247.
[51] *Health Risks of Radon and Other Internally Deposited Alpha-Emitters.*
[52] Fetter and von Hippel, "The Hazard from Plutonium Dispersal by Nuclear-warhead Accidents."

The Urban Case

Suppose a category 3 accident takes place at some airforce base or cantonment, which happens to be at the edge of a major city like Delhi or Karachi or Lahore. These cities will, within the next few years, have a population of about 10 to 12 million people, in an area of about 600 to 800 km^2. Let us consider the worst-case scenario where such a city happens to be downwind at the time of the explosion and has an area large enough for all the plutonium to be deposited.

Note that using a mixing height of H = 300 meters, a wind velocity of u = 1 m/sec and a deposition velocity of v = 0.01 m/sec in equation (2) gives a contamination range L of about 30 km, which is about the width of our assumed cities.[53]

For such cases one can use the uniform density formula equation (7) with a population density P_0 of 15,000 people per km^2 or 1.5×10^{-2} /m^2. It is widely accepted that a single warhead contains a few kilograms of plutonium. Given the estimates cited earlier that about 20% of the plutonium is converted to respirable aerosol, we assume that $Q_0 \approx 1$ kg = 10^6 mg. Using the breathing parameters suggested by the ICRP, we take the age weighted average breathing rate for a South Asian population to be $b = 3.3\times10^{-4}$ m^3/sec.[54] We take the aerosol deposition velocity v to be 0.01m/sec. Inserting these values into equation (7) gives the total amount of plutonium inhaled by the population:

$$M_u = \frac{bQ_0P_0}{v} = 495\,mg$$

This number represents just the effect of plutonium inhalation during the passage of the plume. In addition, there will be further contamination from inhaling plutonium that is resuspended from what was initially deposited on the ground during plume passage. It is simply not feasible to decontaminate or evacuate a major South Asian city. What is likely is that there will be panic and flight creating an unprecedented disaster in its own right. However, in the context of subcontinental urban poverty, most people are in fact likely to

[53] This compares well with experiments at the Nevada Test Site where it was found that "contamination (to a level of significantly residual hazard for which something must be subsequently done) certainly extends for ten miles or more in a downwind direction from ground zero." Chanin and Murfin, "Site Restoration: Estimation of Attributable Costs From Plutonium-Dispersal Accidents."

stay on or return within a short period of time to their original homes. Therefore, we have to multiply the number of deaths estimated above with the resuspension factor in equation (10). We use the values $K_o = 10^{-5}$ m^{-1}, s = 1.96 (ln 2) $years^{-1}$ derived from the leading term in the resuspension formula suggested by the U.S. Nuclear Regulatory Commission in1990.[55] We also use v = 0.01 m/sec to obtain a total inhalation amount of:

$$(M_{tot})_u = 495 \,.\, [\, 1 \; + \; (10^{-5}) \, (0.01) \, (3.15 \times 10^{7)} / \, (1.96 \,)(\ln 2) \,]$$

$$\approx \mathbf{1640} \; \boldsymbol{mg}$$

The final step is to get an estimate of cancer fatalities from such a dosage of inhaled plutonium. As discussed in the previous section, estimates range from 2.85 to 12 cancers/mg of inhaled plutonium. To be conservative, we use 3 cancer deaths per mg of plutonium inhaled, close to the lower limit of what is plausible. Therefore, the number of cancer deaths in this scenario is:

$$N = 1640\, mg \times 3 \; cancer\, deaths / mg \;\; = 4920$$

This has been a conservative estimate. The BEIR and Fetter and von Hippel estimates for fatal cancers/mg would suggest four times as many deaths. It must be remembered that these 5000 deaths (and possibly more) result not from a nuclear explosion with a fission yield, but only from the "worst case" of a category 3 accident, i.e., the detonation of the chemical high explosive in the weapon leading to plutonium dispersal.

A Semi-Urban Case

The urban case estimate above hypothesizes the detonation of the HE in a nuclear weapon in the immediate vicinity of (or inside) a major metropolis like Delhi or Lahore. It may be argued that nuclear weapons are unlikely to be deployed on missiles or bombers so close to a major metropolis. Actually there *are* military bases and

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[54] ICRP, *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 4 Inhalation Dose Coefficients*.

[55] *Severe Accident Risks: An Assessment of Five Commercial Nuclear Power Plants* (Washington, D.C.: U.S. Nuclear Regulatory Commission, report # NUREG-1150, 1990), reproduced in Chanin and Murfin, "Site Restoration: Estimation of Attributable Costs From Plutonium-Dispersal Accidents."

cantonments in the subcontinent right next to big cities. Nevertheless, let us also estimate the damage done in a scenario where a category 3 accident takes place in a military base, at some distance from any city. We consider a case wherein there is a middle-sized city within 50 km downwind from the site of the accident. Jallandhar, Agra and the twin cities of Rawalpindi/Islamabad are examples of such cities.

For this situation schematically sketched in fig.2, there will be plutonium absorption by both the people in that city and the people in the rest of the rural countryside. These two contributions will be additive. For the former we can apply the result in equation (8) reproduced here for convenience:

$$M = M_u \frac{\theta_c}{\theta}\left[e^{-R/L} - e^{-(a+R)/L}\right]$$

When resuspension effects are included for the same reasons as in the worst case example, this amount gets enhanced by the same resuspension factor in equation (10):

$$M_{tot} = M\left[1+\frac{vK_0}{a}\right] = \left(M_u\right)_{tot}\frac{\theta_c}{\theta}\left[e^{-R/L} - e^{-(a+R)/L}\right] \qquad (11)$$

where $(M_u)_{tot}$ gives the mass of plutonium inhaled by a population of uniform density, including the effects of resuspension, that was obtained earlier. For the medium sized city that we are considering, assuming a population of 0.75 million people in an area of 10km ×10km, the population density will be half of the 15000 people per km^2 that was used in the earlier scenario. Therefore, $(M_u)_{tot}$ will be halved to 820 mg.

If we take this middle sized city to be at a distance of 50 km, it will subtend an angle θ_c = 10/50 = 0.2 radians at the point of explosion. The wedge angle θ in the wedge model can take values between 0.1 and 0.3. Let us take the mean of these numbers and use θ = 0.2. The width of the city is r =10km. Lastly the range L is given by Hu/v. Taking a wind velocity u=1 m/sec (≈2 miles per hour), and typical values for the mixing height of H= 500m, and the deposition velocity v = 1 cm/sec, yields L = 50km. This shows that it is not at all unreasonable that winds carry the plutonium aerosol to distances of the order of 50km. Inserting these values into the above equation we get for the total plutonium breathed in by the population of that city:

$$M_{tot} = \frac{(M_u)_{tot}}{e}\left[1 - e^{-0.2}\right] = \frac{820}{e}\left[1 - e^{-0.2}\right] \approx 55mg$$

There is also the plutonium inhaled by the people in the countryside. To estimate this we can use the uniform density result obtained earlier in the "worst case" context, but vastly scaled down by an assumed rural population density of about 200 people per km^2 instead of the 15,000 assumed in the metropolis. This gives for rural inhalation a value of 1640×200/15,000 = 22 mg. Along with the urban contribution obtained earlier of 55 mg, this gives a total of 77 mg, which translates, using the same risk rate of 3 cancer deaths per mg into a total of about 230 fatalities. Although far less than in the earlier example, this number is comparable to major air crash disasters. Again, using the BEIR and Fetter and von Hippel risk estimates for fatal cancers/mg would lead to about four times as many casualties.

In addition to the fatalities, there will be the medical costs of treating the fatal and non-fatal cancers resulting from inhalation of plutonium as well as the cleanup costs for the immediate neighbourhood of the explosion where the contamination levels would be very high.

6. DISCUSSION

We have described a tractable model of plutonium dispersal in the event of a nuclear weapon accident in which the high explosive in the weapon detonates. This was followed by a quantitative discussion of the health hazard posed by inhaled plutonium in terms of potential cancer deaths. We then applied these results to hypothetical accidents of this type in India or Pakistan to get an estimate of nearly 5000 cancer fatalities caused in a worst case scenario of this type. To this human cost has to be added the massive financial cost of even limited decontamination of just the immediate neighbourhood of the accident, which could be at least hundreds of crores of rupees.[56] This would clearly be a major catastrophe.

[56]Chanin and Murfin, "Site Restoration: Estimation of Attributable Costs From Plutonium-Dispersal Accidents."

Should such a category 3 plutonium dispersal accident take place in South Asia, we believe our estimates of damage are quite conservative. We are well aware that the phenomenon of plutonium dispersal and its health hazards is exceedingly complex and that there is considerable variation in the models and parameters used to characterise its different facets. Nevertheless, given the gravity of the problem, one cannot shirk from obtaining at least an order of magnitude estimate of the overall damage. To be useful, this should be done in a manner that is transparent, in both its assumptions and its methodology. This permits any scientific disagreement about the conclusions to focus on individual contributory factors.

In this spirit, we have used the wedge model since it captures the essential physics of plutonium dispersal in simple analytical fashion. It is easy to apply to different situations and the dependence of its results on different contributing factors is easy to pinpoint. While the simplicity of the model means that it cannot reliably give detailed distributions of plutonium contamination, especially in the high-dosage region near the accident site, it is known to give results in agreement with the results of more detailed models and numerical codes at larger distances. In the no-threshold linear model of radiation induced cancer risks, which is generally accepted, it is the low-dosage contamination over large areas that contributes most to cancer fatalities. If, however, there turns out to be a threshold radiation dose below which radiation induced cancer risk vanishes, then the number of fatalities would be reduced. But, in the absence of concrete evidence for a threshold, caution demands that, like BEIR and ICRP, public health risk estimates be made using a no-threshold model.

The wedge model offers a good technique for making an order-of-magnitude estimate of the total number of cancer fatalities from such accidents. Even within the wedge model some of the input parameters like the aerosol deposition speed v, the mixing layer height H, the wedge angle θ can have widely varying values. The biological factors that determine the cancer risks of plutonium inhalation also vary in the literature, depending on the basic data used to arrive at them. We have taken care to point out in this article the range of likely values of all these different parameters, and used conservative values. Admittedly, if in a given incident all the parameters conspire to take extreme

values they can upset even order of magnitude estimates. Notice, however, that the crucial formula (equation 7) for mass of inhaled plutonium in the uniform population density case which we have used for our worst case estimate is independent of most of the uncertain parameters.

It may be argued that the worst case estimate of 5000 (or perhaps as many as four times more) predicted cancer deaths could take a few decades to occur and that they will form only a small fraction of all cancer fatalities during that period due to other causes. But 5000 deaths are still 5000 deaths. That they happen quietly over decades among a largely unsuspecting public does not mitigate the tragedy. If it can be avoided, it must be.

Accidental Nuclear Detonation

Finally let us re-iterate that our estimate of casualties is not for a nuclear explosion, but only for the detonation of the high explosive in the weapon. This category of accidents has been the main theme if this article. But it is worth remembering that the 4th category in our list of possible accidents, wherein the detonation of the high explosive triggers in turn the detonation of the nuclear weapon, is not ruled out. In the history of nuclear weapons development in the U.S. and the Soviet Union, the risks of an accidental nuclear detonation has been a matter of serious concern. These led to the search for several safety mechanisms. In the U.S. particular importance was given to develop nuclear weapon designs that were "one-point safe", i.e., weapons that even though fully assembled would not produce a nuclear yield if the HE were detonated at a single point.[57]

A detailed discussion of weapon system designs is beyond the scope of this paper. Suffice it to say that it has taken considerable testing and re-designing to enhance safety. The U.S. is estimated to have carried out about 130 very low yield safety related tests, of

[57] One point safety requires that the nuclear weapon design inherently, i.e., without any external devices, have a probability of less than one in a million of producing a nuclear yield greater than four pounds of TNT equivalent in the event of a detonation at any one point in the HE system. See Drell and Peurifoy, "Technical Issues of a Nuclear Test Ban," p. 297.

which 62 are officially acknowledged as one-point safety tests.[58] The USSR reportedly conducted about 25 safety tests involving 42 weapons, between 1949 and 1990.[59]

It is in the face of this history that we have to assess the status of nuclear weapon safety in the subcontinent. Given that officially there have been only two sets of tests by India and one by Pakistan, it is reasonable to be concerned about category 4 accidents also. Should such an accident take place, the nuclear yield could be as large as the design yield of the nuclear bomb or warhead.

An accidental nuclear explosion with a yield of 15 kilotons, the same as the weapon detonated over Hiroshima, would destroy over 5 square kilometres from the combined effects of blast damage and firestorms. Over 24 square kilometers would be subject to radioactive fallout at levels such that half the adult, healthy population would die from radiation sickness. If this were to happen in the vicinity of a large South Asian city, several hundreds of thousands of people would die.[60] In addition, such an explosion, especially in times of crises, might be assumed to be a nuclear attack and lead to a nuclear response. Thus an accidental nuclear explosion may even initiate a nuclear war.

In conclusion, even if such a catastrophic accidental nuclear explosion does not occur, we have seen that just the dispersal of plutonium due to a high explosive detonation could lead to several thousand fatalities. Thus, prudence, if nothing else, dictates that India and Pakistan not deploy nuclear weapons. They should also store them far away from missiles and aircraft carrying potentially explosive fuel. A further level of safety may be gained by keeping the weapons disassembled, so that the HE is not close to the fissile material pit. All these steps would not only reduce the danger of accidental explosions, but also reduce the risk of a nuclear weapon being launched through error, panic or miscalculation.

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[58] Thomas B. Cochran and Christopher E. Paine, "Hydronuclear Testing and The Comprehensive Test Ban: Memorandum to Participants JASON 1994 Summer Study," Natural Resources Defense Council, Washington D.C., 1994, p.11.

[59] Robert S. Norris and William Arkin, Soviet Nuclear Testing, August 29, 1949 – October 24, 1990, *The Bulletin of the Atomic Scientists*, May/June 1998.

[60] M.V.Ramana, *Bombing Bombay: Effects of Nuclear Weapons and a Case Study of a Hypothetical Explosion* (Cambridge, U.S.A.: International Physicians for the Prevention of Nuclear War, 1999), p. 31.

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Appendix I: ICRP Model for Plutonium Deposition and Subsequent Behaviour

The principal risk from exposure to plutonium is by inhalation. Calculation of the dose following inhalation can be made by applying the lung model developed by the International Commission on Radiological Protection.[61] In this model, the respiratory tract is represented by five regions, from the nose and mouth down through the alveoli in the lung, where the gas exchange takes place. Once inhaled, the plutonium is quickly distributed between these regions. The fractions of inhaled aerosols, assumed to be log-normally distributed with a median size of 1 micron, deposited in the different regions I-V vary with age and are given in Table 3.

Table 3: Deposition of Inhaled Aerosols in Respiratory Tract Regions

Deposition in Regions	Fraction (%) Deposited at Age		
	3 months	10 years	Adult (male)
I	20.97	17.75	14.89
II	27.20	22.86	18.97
III	1.04	1.17	1.29
IV	2.05	1.70	1.95
V	8.56	9.51	11.48
Total	59.82	52.99	48.58

All the material deposited in the first region goes back to the environment through extrinsic means, such as nose blowing. In the other regions, material is cleared by the movement of particles towards the gastrointestinal tract and lymph nodes, and absorption into body fluids. This absorption depends on the solubility of the plutonium, and as mentioned earlier, the ICRP used Types S, M, and F to denote slow, medium and fast absorption. These processes are described by the clearance rates:

$$\frac{dR_i(t)}{dt} = -\lambda_i(t)R_i(t) + m_{ji}(t)R_j(t)$$

[61] ICRP, *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 4 Inhalation Dose Coefficients*.

where $R_i(t)$ is the amount of material retained in region i (I-V) at time t after intake, $\lambda_i(t)$ is the overall instantaneous rate of clearance of material from region i, and $m_{ji}(t)$ is the rate of clearance of material from any region j into region i. To simplify calculations, the time dependence of $\lambda_i(t)$ is modelled by partitioning each region into multiple compartments. Each compartment is assumed to clear at a constant fractional rate, such that the overall clearance approximates the required time-dependent behaviour.

Region II is divided into two compartments II-A and II-B. About 99.95% of the material deposited in Region II (R_{II}) goes into II-A, which is cleared into the gastrointestinal (GI) tract at the rate of 100/day. The remaining 0.05% goes into II-B, and is cleared to the extrathoracic lymph nodes at a rate of 0.001/day. Region III is divided into three compartments. 49.3% of the material deposited (R_{III}) goes into compartment III-A, that is cleared to compartment II-A at a rate of 10/day; 50% goes into compartment III-B and is cleared to II-A at the rate of 0.03/day. The remaining 0.7% goes into III-C, which clears into the thoracic lymph nodes at the rate of 0.01/day. Of the material deposited in Region IV, i.e., R_{IV}, 49.3% goes into the first compartment IV-A and is cleared into III-A at the rate of 2/day; 50% goes into the second compartment IV-B that is cleared into III-A at the rate of 0.03/day. The remaining 0.7% goes into a third compartment IV-C that is cleared into the thoracic lymph nodes at the rate of 0.01/day. The material deposited in the alveolar region (R_V) is divided into three compartments in the ratio of 3:6:10. The first compartment clears into IV-A at the rate of 0.02/day. The second compartment clears into IV-A at the rate of 0.001/day. The third compartment clears into IV-A at the rate of 0.0001/day and into the thoracic lymph nodes at the rate of 0.00002/day.

Since PuO_2 is fairly insoluble, it is assigned to Type S. Except in the outermost region I, of the plutonium in any compartment, 0.1% is absorbed with a half time of 10 minutes and the remaining 99.9% with a half time of 7000 days. Since the clearance rates from Regions II, III and IV are much faster, there is little absorption from these regions. About 10% of the material deposited in Region V eventually reaches body fluids. Most of the deposited material that is not absorbed is cleared to the GI tract by particle transport.

The small amounts transferred to the lymph nodes continue to be absorbed into body fluids at the same rate as in the respiratory tract, i.e., with a time constant of 7000 days.

Upon leaving the lungs, the plutonium is distributed by the blood. A complicated biokinetic model describes this behaviour. Briefly, this model treats the blood as a uniformly mixed pool that loses activity with a half-time of 0.75 days. It is assumed that the activity in the blood is relatively rapidly exchanged with a "soft-tissue" compartment (ST0), which receives about 30% of the plutonium leaving the blood. The removal half time from ST0 back to the blood is about 1 day. Physiologically ST0 is associated with extra-cellular fluids.[62] Of the plutonium leaving the blood, 80% is deposited in the liver and bone surfaces. Division of this amount between bone and liver varies with age. For babies less than 1 year, the bone receives 87.5% and the liver receives the remaining 12.5%. For children less than 15, the bone receives 75% and the liver receives 25%. For adults, the fractions are 62.5% for bone and 37.5% for the liver. From the bone, the plutonium eventually returns to the blood via bone marrow. The bulk of the plutonium deposited in the liver returns to the blood but a small fraction is expelled to the gastro-intestinal system. A small fraction of the plutonium in the blood is also transferred to the GI system. From the GI system, the plutonium is excreted with faeces. Similarly a small fraction of the plutonium in the blood is transferred to the kidneys and is eventually expelled with urine.

This model allows us to determine the amount of plutonium and its residence time within each region of the respiratory tract. This enables us to calculate an "equivalent dose" (E) to the lung, defined as the product of the activity of the radionuclide, the energy of the radiation emitted, the weighting factor for that type of radiation,[63] the yield of the radiation in question per nuclear disintegration and the fraction absorbed, divided by the mass of the lung. There are similar models for each organ and tissue in the body. This allows one to calculate an effective whole-body dose, defined as:

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[62] See R. W. Leggett, "A Retention-Excretion Model for Americium in Humans," *Health Physics* 62, no. 4 (April 1992) pp. 288-310.

[63] The weighting factor is also called the relative biological effectiveness (RBE); RBE is defined as the ratio of doses from two different radiations that produce equal levels of biological damage in the same biological system; i.e. RBE = dose of reference radiation/dose of test radiation for same level of effect. The reference radiation chosen is usually 250 kV X-rays or Cs-137 or Co-60 gamma rays.

$$E = \sum_T w_T H_T$$

where w_T is the weighting factor or fractional risk factor assigned to the tissue or organ T and H_T is the equivalent dose for that tissue or organ. The weighting factors are given in Table 4.

Table 4: Tissue weighting factors

Organ or Tissue (T)	Weighting Factor (w_T)
Gonads	0.20
Bone Marrow	0.12
Colon	0.12
Lungs	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone Surface	0.01
Remainder	0.05

This effective whole-body dose per unit of activity (Bq) gives the effective dose coefficients shown in Table 2.

While plutonium-239 decays primarily through alpha decay, it also has a weak gamma decay. Therefore, in addition to the internal dose due to inhalation, there could also be an external dose. However, it can be estimated that the external dose from standing in a cloud of plutonium aerosol is a factor of 100 million smaller than the inhalation dose. Similarly, it would take nearly 8000 years for the integrated ground dose (i.e., external dose from plutonium deposited on the ground from a passing cloud of plutonium aerosol) to equal the dose from inhalation.[64]

A special contribution could occur if the skin is punctured or damaged by acid or thermal burns. Under such circumstances a significant amount of plutonium can be absorbed into the subcutaneous tissue and blood. Insoluble particles and metal slivers

deposited below the level of the epidermis are slowly cleared to regional lymph nodes.[65] Investigations involving beagles found that several years after a subcutaneous implant of PuO_2 and $Pu(NO_3)_4$, 33 and 30% respectively of the plutonium was still present in the body, distributed in the injected region, regional lymph nodes, the liver and the skeleton.[66] In our calculation, we will not include this contribution since it is not generalizable to all those exposed. But in the case of people with wounds or burns exposed to plutonium, this could be significant.

Appendix II: Health Effects of High Doses of Plutonium

There are specific health effects associated with inhalation of comparatively large amounts of plutonium or other transuranic compounds. There are no data involving large deposits of plutonium in people. Experiments with animals have shown radiation induced pneumonitis (inflammation of the lung) and fibrosis (scarring of lung tissue).[67] Diffuse fibrosis, following deposition of 37-74 kBq/g of lung tissue of plutonium, led to reduced efficiency of the respiratory system and death within a month or two.[68] Deposition of much smaller amounts, about 3.7 kBq/g of lung of plutonium-239 in the alveolar region of the lung, have been shown to lead to respiratory failure within 10 months in dogs. Even smaller amounts of alveolar deposition, about 1.85 kBq/g of lung tissue, have induced, *inter alia*, pulmonary fibrosis and early tumour formation within 5 years.[69]

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[64] Fetter and von Hippel, "The Hazard from Plutonium Dispersal by Nuclear-warhead Accidents."
[65] *Health Risks of Radon and Other Internally Deposited Alpha-Emitters*, p. 307.
[66] G. E. Dagle and C. L. Sanders, "Plutonium-induced Wounds in Beagles," *Health Physics*, Vol. 47, (1984) pp. 73-84.
[67] G. E. Dagle and C. L. Sanders, "Radionuclide Injury to the Lung," *Environmental Health Perspectives*, Vol. 55, (1984) pp. 129-137.
[68] International Commission on Radiological Protection (ICRP), *Recommendations of the International Commission on Radiological Protection*, ICRP Publication 26, (Oxford: Pergamon, 1977).
[69] International Commission on Radiological Protection (ICRP), *Biological Effects of Inhaled Radionuclides*, ICRP Publication 31, (Oxford: Pergamon, 1980).